Investigative Ophthalmology & Visual Science



*Invest Ophthalmol Vis Sci* 2012;53: E-Abstract 4378.




**4378—A604**

# The Relationship Between *CFH* And *ARMS2* Genotypes and Neuroretinal Function In Persons Without Age-Related Macular Degeneration

**Beatrix K. Feigl[1,2], C Phillip Morris[1] and Andrew J. Zele[1]**

[1]Institute of Health & Biomedical Innovation, Queensland University of Technology, Brisbane, Australia
[2]Queensland Eye Institute, Brisbane, Australia

**Commercial Relationships: Beatrix K. Feigl**, None; **C Phillip Morris**, None; **Andrew J. Zele**, None

**Support:** QUT Vice-Chancellor Fellowship

## Abstract

**Purpose:**To determine whether neuroretinal function differs in healthy persons with high and low-risk gene variants for age- related macular degeneration (AMD) and no ophthalmoscopic signs of AMD, and to compare those findings in persons with manifest early AMD.

**Methods:**Neuroretinal function was assessed with the multifocal electroretinogram (mfERG) (VERIS, Redwood City, CA,) in 32 participants (22 healthy persons with no clinical signs of AMD and 10 early AMD patients). The 22 healthy participants with no AMD had high or low-risk genotypes for either the *CFH* (rs380390) and/or *ARMS2* (rs10490920). We used a slow flash mfERG paradigm (3 inserted frames) and a 103 hexagon stimulus array. Recordings were made with DTL electrodes; fixation and eye movements were monitored online. Trough N1 to peak P1 response densities (N1P1-RD) and P1-implicit times (P1-IT) were analysed in 5 concentric rings.

**Results:**N1P1 response densities (mean ± SD) for concentric rings 1-3 were on average significantly larger in participants with high-risk genotypes (ring 1: 17.97 nV/deg$^2$ ± 1.9, ring 2: 11.7 nV/deg$^2$ ±1.3, ring 3: 8.7 nV/deg$^2$ ± 0.7) compared to those with low-risk genotypes (ring 1: 13.7 nV/deg$^2$ ± 1.9, ring 2: 9.2 nV/deg$^2$ ±0.8, ring 3: 7.3 nV/deg$^2$ ± 1.1) and compared to persons with early AMD (ring 1: 15.3 nV/deg$^2$ ± 4.8, ring 2: 9.1 nV/deg$^2$ ±2.3, ring 3 nV/deg$^2$: 7.3± 1.3) ($p<0.5$). The group implicit times, P1-ITs for ring 1 were on average delayed in the early AMD patients (36.4 ms ± 1.0) compared to healthy participants with high-risk (35.1 ms ± 1.1) or low-risk genotypes (34.8 ms ±1.3), although these differences were not significant.

**Conclusions:**Neuroretinal function in persons with normal fundi can be differentiated into subgroups based on their genetics. Increased neuroretinal activity in persons who carry high-risk AMD genotypes may be due to genetically determined subclinical inflammatory and/or histological changes in the retina. Assessment of neuroretinal function in healthy persons genetically susceptible to AMD may be a useful early biomarker before there is clinical manifestation of AMD.

**Keywords:** age-related macular degeneration • electrophysiology: clinical • aging